

VENOUS THROMBOEMBOLISMS

A Guide to Recognition, Treatment, & Care of Pulmonary Embolisms and
Deep Vein Thrombosis

Objectives

- Review the incidence, risk factors, and causes for deep vein thrombosis (DVT) and pulmonary embolism (PE)
- Recognize the pathophysiology and signs and symptoms of DVT and PE
- Discuss diagnosis, treatment, and prevention of DVT and PE
- Identify complications related to DVT and PE
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Venous Thromboembolisms

- Venous thromboembolisms (VTEs) are blood clots that start in a vein
- Third leading vascular diagnosis in the United States
- Affects 300,000 to 600,000 Americans every year
- Most commonly occurs in adults over 60 years old
- Two types of VTEs
 - Deep vein thrombosis (DVT): clot in a deep vein; most often in the leg but can also occur in the arms or other veins
 - Pulmonary embolism (PE): a DVT that has dislodged from the vein wall and traveled into the arteries of the lungs blocking all or some of the blood flow; DVTs originating in the thighs are most likely to break off and become PEs

VTE Statistics

- Annual average number of hospitalizations where VTE diagnosed
 - Aged 18-39 years: 54,034
 - Aged 40-59: 143,354
 - Aged 60 years or older: 350,208
- Annual average number of VTE diagnoses per sex
 - Males: 250,973
 - Females: 296,623
- Thromboembolism rates are higher in African Americans than in whites and are lowest in Asians
- Although females experienced more total VTEs, PEs are more common in males than in females

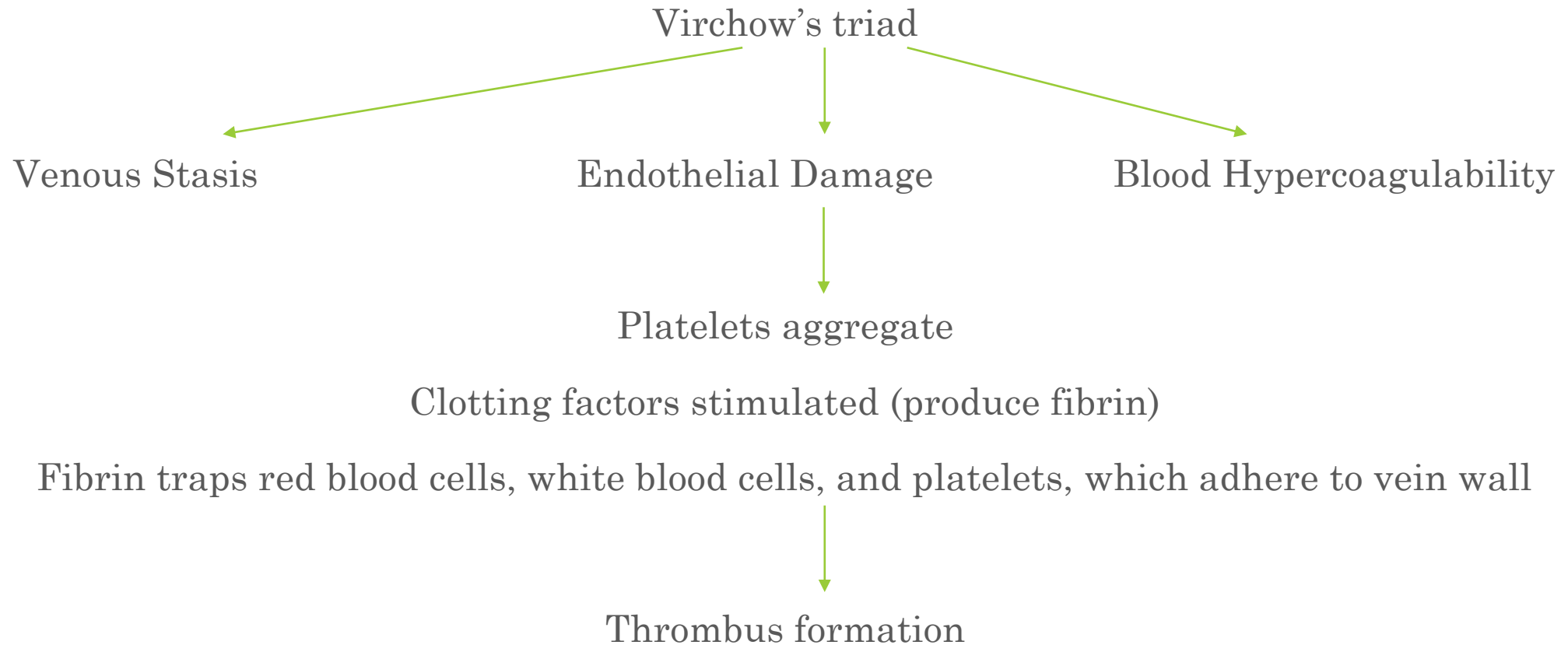
Prognosis

- Death from VTE accounts for 5-10% of all deaths in the hospital
- Approximately one third of PE cases are fatal
 - 67% are not diagnosed ante mortem
 - 34% occur rapidly
- High rate of clinically unsuspected DVTs and PEs causes therapeutic and diagnostic delays accounting for significant morbidity and mortality
- Risk is reduced by 95% with proper treatment of thromboembolic disease
- Once treatment is discontinued, there is a risk of reoccurrence related to compliance and total number of risk factors
 - 5-7% of all reoccurrences are fatal

Virchow's Triad

- Combination of three factors cause VTEs
 - Venous stasis: dysfunctional valves or inactive muscles in the extremities
 - Can occur in people with: pregnancy, chronic heart failure, atrial fibrillation, extended travel (e.g. flights and car rides), obesity, prolonged surgical procedure, immobility
 - Damage of the endothelium: stimulates platelet activation which initiates coagulation cascade
 - Direct: surgery, intravascular catheterization, trauma burns
 - Indirect: chemotherapy, diabetes, sepsis
 - Hypercoagulability of vein: vein more likely to form thrombus
 - Occurs in disorders: anemia, polycythemia, malignancies, nephrotic syndrome, hyperhomocysteinemia, protein C and S deficiency
 - Can be caused by medication: corticosteroids, estrogens
 - Women who use oral contraceptives and smoke cigarettes, are over 35 years old, and have family history of VTEs
- Patients who develop VTEs typically have predisposing factors for the three components

Virchow's Triad Pathophysiology



Pulmonary Embolisms

(PEs)

Pathophysiology of PEs

- Venous thrombi most often originate in deep venous valve pockets and other sites with venous stasis
 - The deep venous system of lower extremities most common, but thrombi can also form in the upper extremity, pelvic, or renal veins or the right heart chambers
- The thrombi travel through the vascular system into the right atrium and right ventricle, where it can then enter the pulmonary vasculature and become lodged, blocking blood flow
- They typically lodge in the bifurcation of the main pulmonary artery or the lobar branches
- Causes hemodynamic compromise

Symptoms of PEs

- Classic presentation: sudden onset of pleuritic chest pain, shortness of breath, hypoxia
- Most commonly patients present with either sudden hemodynamic collapse or progressive dyspnea
- PEs should be suspected in all patients with undiagnosed respiratory problems
- Abnormal symptoms include: seizures, syncope, abdominal pain, fever, productive cough, wheezing, decreasing level of consciousness, atrial fibrillation, hemoptysis, flank pain, delirium

Assessment Signs of PEs

- Dyspnea (most common sign occurring in 85% of cases)
- Tachypnea
- Rales, wheezes
- Accentuated second heart sound
- Tachycardia of over 100 beats per minute
- Fever greater than 100.4 degrees F
- Diaphoresis
- S3 or S4 gallop
- Heart murmur
- Cyanosis

Risk Factors and Causes of PEs

- Predisposition to thrombus formation called Virchow triad
 - Endothelial injury, stasis of blood flow, blood hypercoagulability
- Often multiple causes at play or no one cause is readily apparent
 - Venous stasis: accumulation of platelets and thrombi in veins
 - Hypercoagulable states: can be altered by disease, obesity, trauma, surgery
 - Immobilization: local venous stasis accumulating clotting factors and fibrin
 - Surgery or trauma: activate clotting factors and cause immobility
 - Pregnancy: fatality from PEs in 1 to 2 cases per 100,000 pregnancies
 - Oral contraceptives or estrogen replacement
 - Malignancy: can occur with solid tumors, leukemias, and lymphomas
 - Hereditary factors
 - Acute medical illness: AIDS, Behcet's disease, CHF, myocardial infarction, polycythemia, lupus, ulcerative colitis
 - Other factors (e.g. drug abuse, hyperlipidemia, IBD, sleep apnea)

Diagnostic Labs for PEs

- Hypercoagulation workups useful if no history or signs of embolic disease
 - Antithrombin III deficiency, protein C or protein S deficiency, lupus anticoagulant, homocystinuria, occult neoplasm, connective tissue disease
- Tests for suspected PE
 - D-dimer, Ischemia-modified albumin level, white blood cell count, arterial blood gases, serum troponin levels, brain natriuretic peptide (BNP)

Imaging Studies for PEs

- Computed tomography angiography (CTA/CT scan): most frequently used, requires contrast media
- Pulmonary angiography: most sensitive and specific, but expensive/invasive
- V/Q scanning: used when contrast media contraindicated
- Chest radiography
- ECG
- MRI
- Echocardiogram
- Venography
- Duplex ultrasonography

Medical Treatments for PEs

- Begin full anticoagulation therapy once PE suspected
- Thrombolytic therapy patient criteria: systolic blood pressure < 90 or clinical suspicion of developing hypotension, absence of high bleeding risk
- Anticoagulation medications:
 - Unfractionated heparin, low-molecular-weight heparin (LMWH), factor Xa inhibitors, fondaparinux, warfarin
 - LMWH recommended choice for nonmassive PEs
- Thrombolytic agents:
 - Alteplase, reteplase

Surgical Treatments for PEs

- Catheter embolectomy and fragmentation or surgical embolectomy
 - Indicated for patients with massive PEs when fibrinolytic therapy is contraindicated
 - High mortality rate, so it should only be used when other options are unusable
- Placement of inferior vena cava (IVC) filter
 - Indicated for patients at high risk of recurrent PE or when anticoagulation therapy is contraindicated
 - IVC filter placed at diaphragm level in inferior vena cava via femoral vein

Potential Complications with PEs

- Mortality rate for massive PE is 30 to 60% with most deaths occurring within one to two hours
- Pulmonary infection most likely when:
 - Large or medium-sized vessel is occluded
 - Insufficient collateral blood flow from bronchial circulation
 - Lung disease
- Pulmonary hypertension
 - Emboli must be massive or PEs recurrent to result in pulmonary hypertension

PE Prevention

- Difficult to prevent outpatient PEs
- Majority of PEs occur in hospitalized patients so prevention should be focused on patients through prophylaxis:
 - Mechanically, heparin, LMWH, warfarin
- QThrombosis algorithm identifies high-risk patients who are currently asymptomatic
 - Estimates risk at one- and five-year points
 - Used to guide prophylaxis and medication use

Deep Vein Thromboses

(DVTs)

Pathophysiology of DVTs

- Majority of DVTs are able to be resolved with no complications
 - Fatalities from DVTs occur when thrombi break from a deep vein and travel to lungs, resulting in a PE
- Thrombus formed through localized platelet aggregation and fibrin entrapment of red blood cells, white blood cells, and more platelets
- Thrombus enlarges by trapping more blood cells and fibrin
- Tail forms on the thrombus, which blocks the vein's lumen
- Endothelial cells will gather on a thrombus if the vein is only partially blocked, subsequently stopping the thrombotic process
- If the thrombus does not detach it undergoes lysis or becomes fully organized within five to seven days
- An organized thrombus that detaches can become a PE

Symptoms of DVTs

- Edema
- Leg pain
- Tenderness
- Warmth of skin around thrombosis

Assessment Signs of DVTs

- Many cases of DVTs are asymptomatic and go undiagnosed
- Homans sign: dorsiflexion of foot causes calf pain
- Palpable, indurated, tender subcutaneous venous segment
- Discoloration of lower extremity
- Blanching of leg due to edema
- Leukocytosis

Risk Factors and Causes of PEs

- Risk factors and causes similar to those seen in PEs because DVTs precede PEs
 - Acute infectious disease, age older than 75 years, cancer, history of VTE
- Immobilization
 - Short term (e.g. long flights or general anesthesia)
 - Long term (e.g. hospitalization for pelvic, hip, spine surgery or paraplegia)
- Decreased venous blood flow due to increased blood viscosity
 - Polycythemia rubra vera (PRV), thrombocytopenia, dehydration
- Increased central venous pressure
 - Mechanical or functional decrease of blood flow to legs
 - Neoplasm, pregnancy, stenosis, congenital anomaly
- Anatomical variant
- Injury to vein

Labs for DVTs

- Routine blood tests help determine which patients are at high risk for DVTs
 - D-dimer assay
 - Levels of antithrombin III (ATIII)
 - N-terminal pro-brain natriuretic peptide (NT-proBNP)
 - C-reactive protein (CRP)
 - Erythrocyte sedimentation rate (ESR)

Medical Treatments for DVTs

- Primary treatment objectives
 - Prevent PE
 - Reduce mortality
 - Prevent postthrombotic syndrome (PTS)
- Anticoagulation therapy is the primary treatment and often is the only treatment needed to break up the DVT
- Outpatient treatment
 - LMWH followed by vitamin K antagonists
- Inpatient treatment
 - LMWH, fondaparinux, unfractionated heparin, warfarin

Outpatient Management for DVTs

- Patients with DVTs are typically able to be treated outside the hospital unless any of the following exclusion criteria are met:
 - Suspect or proven PE
 - Significant cardiovascular or pulmonary comorbidity
 - DVT is in the iliofemoral vein
 - Unable to use anticoagulant therapy
 - Coagulation disorder
 - Bleeding disorder
 - Pregnancy
 - Morbid obesity
 - Renal failure
 - Inability to follow treatment plan properly
 - Homeless
 - Lives too far from hospital

Potential Complications with DVTs

- Asymptomatic PE present in 40% of patients with diagnosed and symptomatic DVT
- Paradoxical emboli
- Recurrent DVT
- Post-thrombotic syndrome
 - Occurs in 20 to 50% of patients even when anticoagulation therapy used properly
 - Caused by chronic venous hypertension, stiff vein walls, persistent venous obstruction
 - Symptoms: pain, aching, heaviness, cramps, itching, tingling
 - Assessment: edema, increased pigmentation, eczema, lipodermatosclerosis
 - Occurs within two years after a DVT
 - May cause venous ulceration

Potential Complications with DVTs (continued)

- Phlegmasia cerulea dolens: swollen, blue, painful leg
 - Rare complication
 - Near total occlusion of venous outflow of major leg veins due to severe DVT
 - Symptoms
 - Sudden, extreme swelling
 - Deep pain
 - Intense cyanosis of affected extremity
 - May result in arterial occlusion, which can cause gangrene and require amputation

Techniques for Preventing DVTs

- Reposition patient every two hours
- Teach patients to flex and extend their feet, knees, and hips every two to four hours unless contraindicated
 - If not contraindicated, nurse should perform these movements on patients who are unable to do so themselves
- If able, patients should walk or get into chair four to six times each day
- Elastic compression stockings
 - Proper use: toe hole under toes, heel patch over heel, thigh gusset on inner thigh, no wrinkles, not rolled down, not cut
- Sequential compression devices (SCDs)
 - Ensure correct fit
 - Wear continuously unless bathing, ambulating, or having skin assessed

Superficial Vein Thromboses

(SVT)

SVTs

- Blood clot in a vein near the surface of the skin
- Assessment
 - Firm, palpable, cordlike vein; itchiness, tenderness, painful to touch, redness, warmth, mild temperature elevation, leukocytosis
- Risk factors
 - Increased age, pregnancy, obesity, malignancy, thrombophilia, estrogen therapy, recent treatment for varicose veins, long-distance travel, history of PE, DVT, or SVT
- Care
 - Duplex ultrasound for diagnosis, LMWH for 45 days or fondaparinux
 - SVT less than 5 cm in the saphenofemoral junction may not need anticoagulation therapy, and only NSAIDs to ease symptoms
- Patient teaching
 - Wear elastic compression stockings and apply topical NSAIDs

Patient Care

For VTEs (PEs and DVTs)

Healthcare-Associated VTEs

- The CDC is focusing on promoting, translating, and implementing strategies to prevent healthcare-associated VTEs (HA-VTE). Prevention strategies are not being consistently used throughout hospitals and other healthcare clinics, which causes premature deaths and costs the U.S. healthcare system \$1 billion per year
1. Strengthen healthcare monitoring of HA-VTE
 - Advance and promote methods and tools to improve and support monitoring of HA-VTE occurrence and prevention
 2. Identify and promote best practices for HA-VTE prevention
 - Identify and share proven prevention tools and resources for partners and stakeholders
 3. Increase education and awareness of HA-VTE
 - Share evidence-based education tools and strategies to increase awareness of risks for and prevention of HA-VTE

(CDC, 2018)

Nursing Diagnoses for VTEs

- Acute pain related to venous congestion, impaired venous return, inflammation
- Risk for impaired skin integrity related to altered peripheral tissue perfusion
- Ineffective health maintenance related to lack of knowledge
- Potential complication: bleeding related to anticoagulation therapy
- Potential complication: pulmonary embolism related to embolization of thrombus

Primary Anticoagulant Therapy

- Vitamin K Antagonists
 - Warfarin
 - Route: PO
 - Therapeutic level lab: INR
 - Antidote: Vitamin K
- Indirect Thrombin Inhibitor
 - Unfractionated heparin
 - Route: Continuous IV, intermittent IV, or subcutaneous injection
 - Therapeutic level lab: aPTT or ACT
 - Antidote: protamine
 - Low-molecular-weight heparin (LMWH)
 - Lovenox, Fragmin
 - Route: subcutaneous
 - Routine coagulation labs not required
 - Antidote: protamine

Additional Anticoagulant Therapy

- Direct Thrombin Inhibitors
 - Hirudin derivatives
 - Lepirudin, desirudin, bivalirudin
 - Route: IV or subcutaneous
 - Therapeutic level lab: ACT or aPTT
 - Used for patients with heparin-induced thrombocytopenia who still need anticoagulation
 - Antidote: none
 - Synthetic thrombin inhibitors
 - Route: IV or subcutaneous
 - Therapeutic level lab: aPTT
 - Used for patients at risk of HIT or who have had joint replacement surgery
 - Antidote: none
- Factor Xa inhibitors
 - Arixtra, Xarelto, Eliquis
 - Route: subcutaneous or PO
 - Therapeutic level lab: routine coagulation tests not required
 - For VTE prophylaxis and treatment
 - Antidote: none

Anticoagulation Medication Labs

Lab	Drugs Monitored	Normal Value	Therapeutic Value
INR: international normalized ratio	Vitamin K antagonists	0.75-1.25	2-3
aPTT: activated partial thromboplastin time	Unfractionated heparin, hirudin derivatives, synthetic thrombin inhibitors	25-35 seconds	46-70 seconds
ACT: activated clotting time	Unfractionated heparin, hirudin derivatives, synthetic thrombin inhibitors	70-120 seconds (varies based on system and test reagent or activator)	> 300 seconds
Anti-factor Xa	LMWH, factor Xa inhibitors	0 U/mL	LMWH: 0.6-1.0 U/mL Factor Xa inhibitors: 0.2-1.5 U/mL

Heparin-Induced Thrombocytopenia (HIT)

- Potentially deadly complication of heparin therapy
- Type 1 HIT: occurs within first two days of heparin therapy
 - Nonimmune disorder caused by heparin's direct effect on platelets
- Type 2 HIT: occurs within four to ten days of heparin therapy
 - Immune-mediated disorder that threatens life and limb
- Suspected when platelet count decreases by 50% of baseline, acute systemic reactions, or skin lesions at injection site
- Generally patient does not experience bleeding, but rather PE or DVT
- More commonly occurs with unfractionated heparin instead of LMWH
- Mortality rate is approximately six to ten percent

Interventions for VTEs

- Focus care on prevention of emboli formation and reduction of inflammation
- Monitor vital signs, arterial blood gases, cardiac rhythm, lung sounds, anxiety
- Monitor labs based on the anticoagulant used
 - INR, aPTT, ACT, anti-factor Xa, CBC, creatinine, factor X levels
- Administer and titrate anticoagulants based on lab results
- Monitor for signs of bleeding
 - Epistaxis, bleeding gums, blood in bodily fluids/stool, oozing from trauma site or surgical site, decreased blood pressure, increased heart rate, mental status changes
- Develop prevention plan
- Provide patient teaching

Patient Care for VTEs

- Injections
 - Intramuscular injections should be avoided in patients receiving anticoagulant therapy, minimize venipunctures, use smaller gauge needles when possible, apply manual pressure as needed on venipuncture sites
- General care
 - Ensure clothing is loose, moisturize skin, avoid straight razors, avoid removing or disrupting established clots, humidify O₂ if oxygen needed, perform oral care gently, reposition frequently, administer stool softeners if patient is straining, lubricate tubes, avoid restraints when possible, use elastic compression or sequential compression devices as ordered, perform fall risk assessment, perform skin breakdown assessment

Patient Teaching for Anticoagulant Therapy

- Provide thorough explanation of purpose of anticoagulants
- Take medication at same time each day
- If required, obtain blood tests to assess effectiveness and therapeutic dose
- Contact emergency medical help for any of the following
 - Blood in urine or stool; black, tarry stool; vomiting blood or “coffee ground” emesis; unusual bleeding from gums, skin, nose, menstrual bleeding; severe headache; severe stomach pains; chest pain; shortness of breath; heart palpitations; weakness; dizziness; altered mental status; cold, blue, or painful feet
- Avoid vigorous oral care, contact sports, straight razors
- Avoid aspirin and NSAIDs
- Wear a medic alert bracelet detailing type of anticoagulant therapy
- Avoid dramatically increasing intake of foods with vitamin K
- Consult with healthcare provider about any medications or supplements
- Inform all healthcare providers and dentists about anticoagulant therapy

Outpatient Teaching

- Focus on modification of VTE risk factors
- Wear elastic compression stockings for at least two years following PE or DVT
 - Supports vein walls, decreases swelling, decreases pain, reduces likelihood of mild post-thrombotic syndrome
- If indicated, teach patient to lose weight and/or stop smoking
- Discourage patient from sitting or standing in motionless, leg-dependent positions
- Instruct patients to move feet and legs when sitting in one place for extended time periods (e.g. plane rides, meetings)

Contraindicated Herbal and Dietary Supplements and Foods

- Patients on anticoagulation therapy may increase their risk of bleeding by using/eating the following:
 - Bilberry, black cohosh, chamomile, chondroitin sulfate, DHEA, feverfew, garlic, ginger, ginkgo biloba, ginseng, goldenseal, green tea, melatonin, niacin, omega-3 fatty acids, psyllium, red yeast rice extract, saw palmetto, soy, turmeric
- Patients taking warfarin should not heavily increase their intake of vitamin K as it will affect INR levels
 - Foods high in vitamin K: leafy greens (e.g. kale, spinach, collards, romaine), Brussel sprouts, cauliflower, cabbage
- All patients on anticoagulation therapy should consult with a healthcare provider before adding any herbal or dietary supplements or altering their diet

PEs and DVTs during Pregnancy

- Greater risk of blood clots during pregnancy because:
 - Blood clots more easily to reduce blood loss during labor and delivery
 - Blood vessels around the pelvis are pressed on by the fetus causing less blood flow to the legs during later pregnancy
- Additional risk factors:
 - Family or personal history of blood clots or clotting disorder, C-section, bedrest, complications during pregnancy or childbirth, certain chronic conditions (e.g. heart or lung condition, diabetes)
- Prevention strategies
 - Educate all pregnant women on signs of DVTs and PEs
 - Ensure patient discusses all possible risk factors with healthcare provider

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